

Hemoptysis and lung disease as a manifestation of pulmonary vein stenosis after cryoballoon catheter ablation for atrial fibrillation

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Atrial fibrillation (AF) is a common and increasingly prevalent cardiac arrhythmia.¹ Recently, new therapeutic options have been introduced, such as catheter ablation. Cryoballoon ablation with pulmonary vein (PV) isolation is a novel technology for the treatment of AF and a good alternative to radiofrequency (RF) ablation. A recent study, Sustained Treatment of Paroxysmal Atrial Fibrillation (STOP AF), has shown that cryoballoon ablation is a safe procedure.³ On the other hand, in RF ablation, such complications as tamponade, stroke, transient ischemic attack, and PV stenosis (PVS) may occur.² The incidence of PVS after cryoballoon catheter ablation was reported to be 3.1%. It was asymptomatic in 60% of cases,³ while a massive occlusion of more than 1 PV, resulting in symptoms or requiring an intervention, was observed only in 0.17% of cases.⁴ Symptoms of PVS include dyspnea on exertion or at rest, aching, burning, or pleuritic chest pain, cough, and hemoptysis.

We present a case of a patient with hemoptysis and lung disease due to PVS after cryoballoon catheter ablation for AF. A 57-year-old man suffering from arterial hypertension was admitted to our pulmonary department with a chronic cough, recurrent hemoptysis, dyspnea, and chest pain. Eight months earlier, the patient was for the first time treated with balloon cryoablation for paroxysmal AF. Before treatment, symptoms of AF occurred a few times a day. During the procedure, an Arctic Front, CryoCath™ (Medtronic, United States) catheter was used and the minimal temperature reached was -65°C. After the procedure, the patient received treatment with rivaroxaban at a daily dose of 20 mg for 3 months. Shortly after the anticoagulant treatment had been discontinued, dry cough, chest pain, and hemoptysis

occurred. The patient was referred for high-resolution computed tomography (CT) by a pulmonologist, which revealed ground glass opacities in segments 6 and 7 of the right lung (FIGURES 1AB). A bronchofiberscopy showed locally swollen mucosa. The results of bacteriological examinations were negative, while laboratory tests were positive for antineutrophil cytoplasmic antibodies (pANCA). Alveolar hemorrhage due to pANCA vasculitis was suspected, and the patient received 16 mg of methylprednisolone daily, but without improvement. After 2 months, his condition worsened.

On admission to our department, we performed a CT pulmonary angiography, which confirmed the complete occlusion of the right lower PV (FIGURES 1CD) and stenosis of the left lower PV. A bronchofiberscopy confirmed the presence of blood in the main bronchi. Bronchoalveolar lavage revealed hemosiderin in 98% of macrophages. Treatment with a therapeutic dose of enoxaparin was started. We did not observe increased bleeding from the bronchial tree. After 3 months of therapy, the patient's clinical condition improved and pulmonary symptoms resolved. A control CT showed substantial regression of lower lobe infiltrates (FIGURES 1E-H). We observed reopening of the right lower PV with 75% residual stenosis of the vessel. The patient still experienced mild dyspnea on exertion, and a cardiologist decided on watchful waiting without a percutaneous intervention. Unfortunately, AF recurred several times within the first year after ablation. The patient remains under the care of a pulmonologist and cardiologist and awaits further tomographic and echocardiographic evaluation of the disease.

In our patient, an accurate diagnosis might have been delayed because of the longer interval

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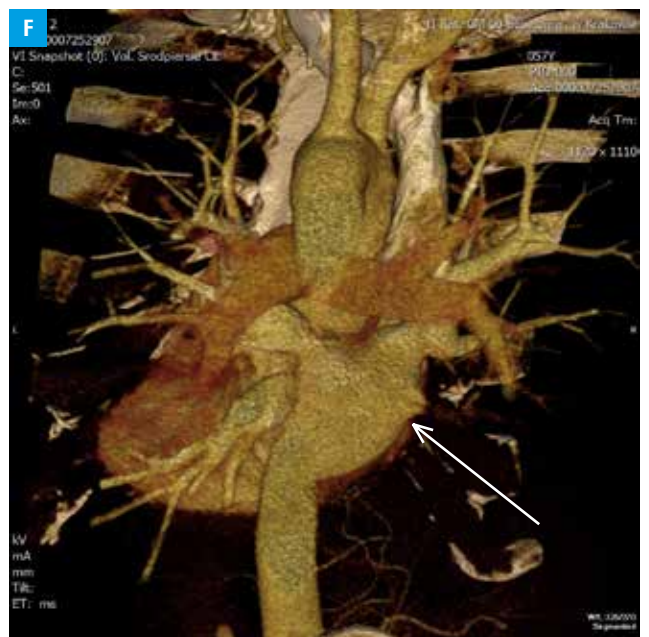
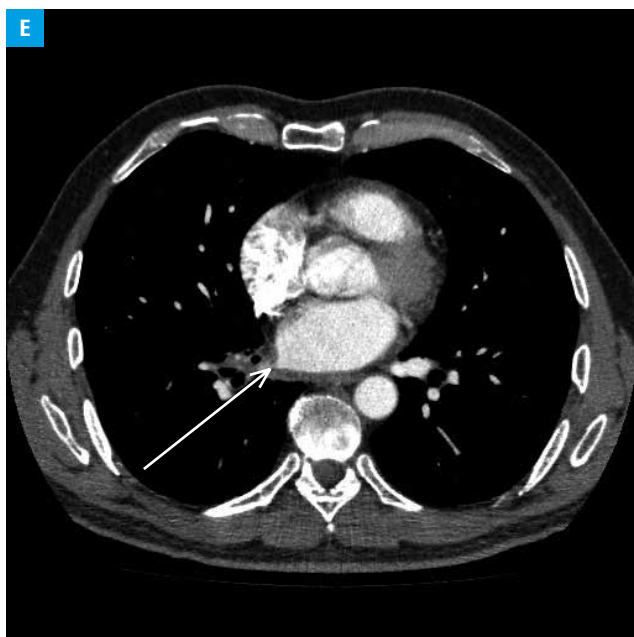
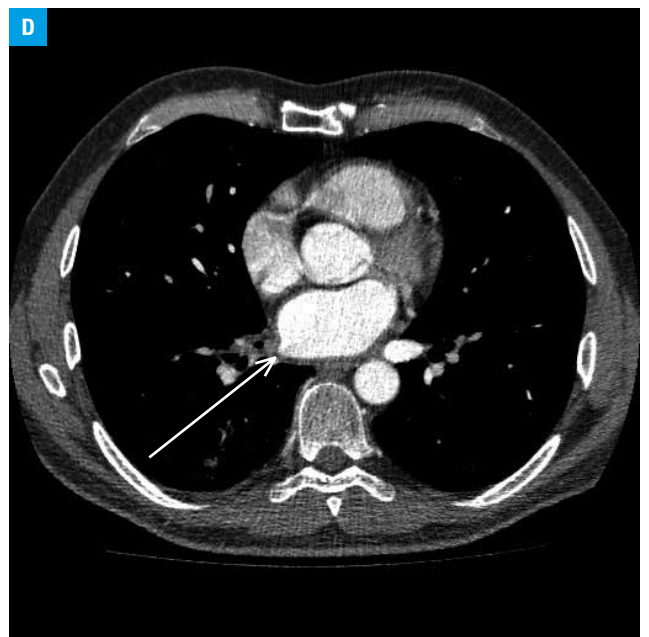
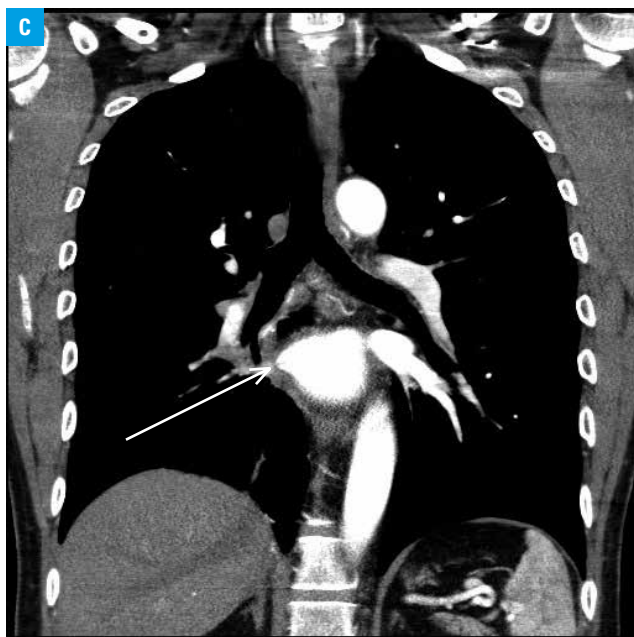
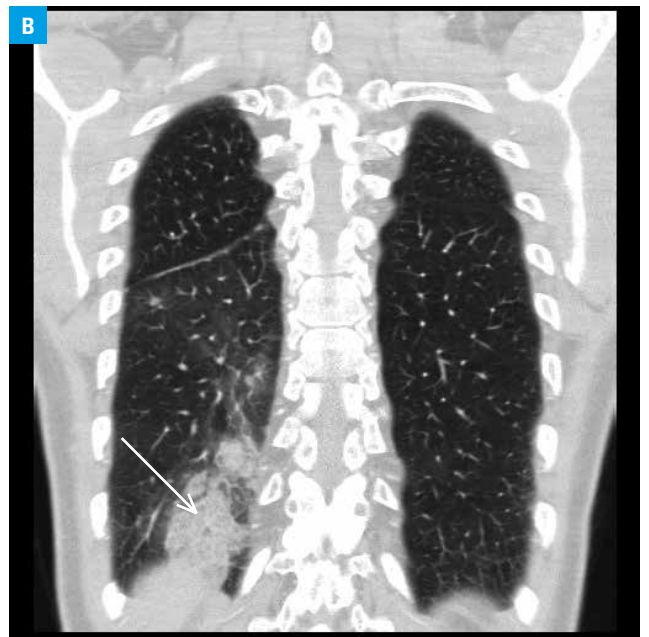
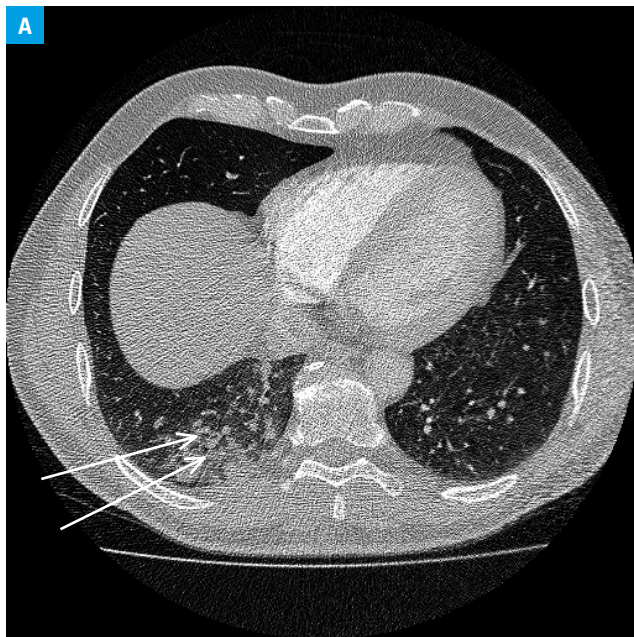
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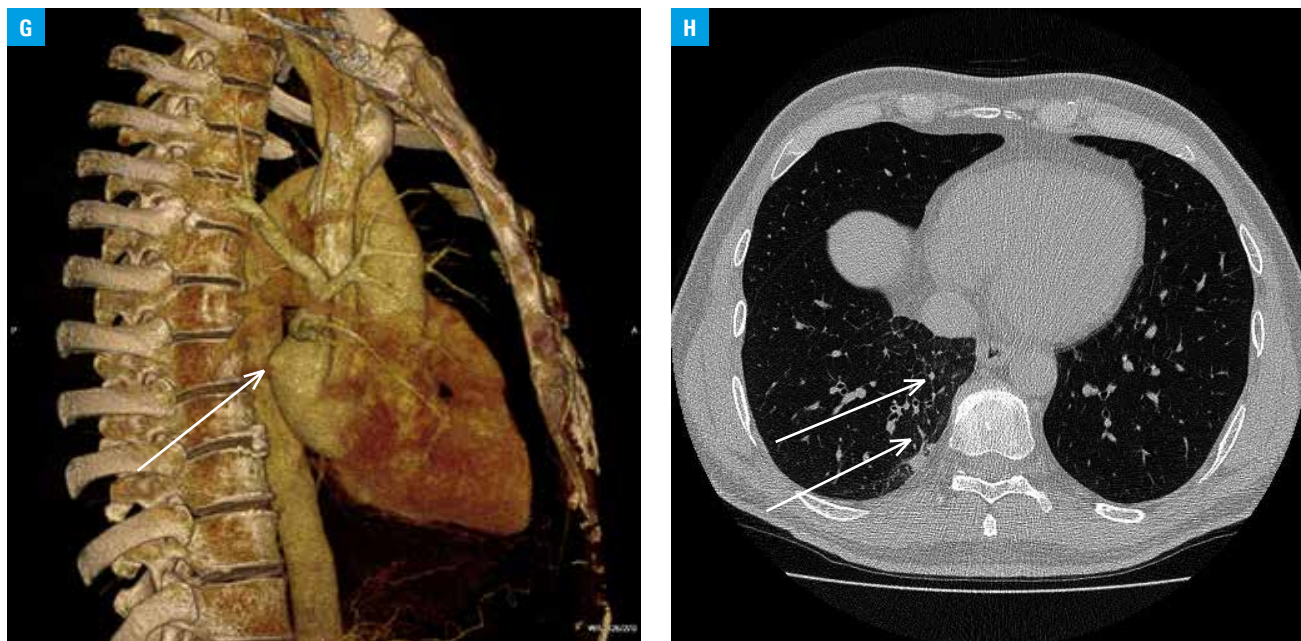


FIGURE 1 **A** – a computed tomography (CT) scan of the lungs; right lower lobe infiltrates with regions of lung infarct (arrows); cross-sections (pulmonary window); **B** – a CT scan of the lungs; right lower lobe lung infiltrates with regions of lung infarct (arrow) (front section); **C** – a CT scan of the lungs; the loss of contrast in the right lower pulmonary vein (front section) (arrow); before treatment with a therapeutic dose of enoxaparin; **D** – a CT scan of the lungs; the loss of contrast in the right lower pulmonary vein (cross-sections) (arrow); before treatment with a therapeutic dose of enoxaparin (arrow); **E** – a CT scan of the lungs; visible partial recanalization of the right lower pulmonary vein after 3 months of treatment with a therapeutic dose of enoxaparin; **F** – a 3-dimensional reconstruction; the loss of contrast in the right lower pulmonary vein before treatment (arrow – left atrium); **G** – a 3-dimensional reconstruction; the loss of contrast in the right lower pulmonary vein before treatment (side view) (arrow – left atrium); **H** – a CT scan of the lungs; a significant regression of consolidations and infiltrates in the area of the right lower lobe after 3 months of treatment with a therapeutic dose of enoxaparin (arrows), cross-sections (pulmonary window)

between the cardiac treatment and pulmonary symptoms. This is the reason why patients are often wrongly diagnosed with pneumonia, pulmonary embolism, lung cancer, or asthma. Pulmonologists may also miss a link between lung disease and hemoptysis and a previous history of AF. A multi-detector cardiac CT or dynamic magnetic resonance imaging for an early diagnosis is the modality of choice. The primary treatment option for extensive PV stenosis and occlusion is PV angioplasty either by catheter dilation or cardiac surgery. In conclusion, although PV stenosis is a rare complication, it should be considered when dyspnea, pulmonary infiltration, or hemoptysis occurs after PV ablation for AF.

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